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The effect of vitamin K₁ supplementation for 12 months on bone mineral density and indices of vitamin K status and bone turnover in adult Crohn’s disease patients

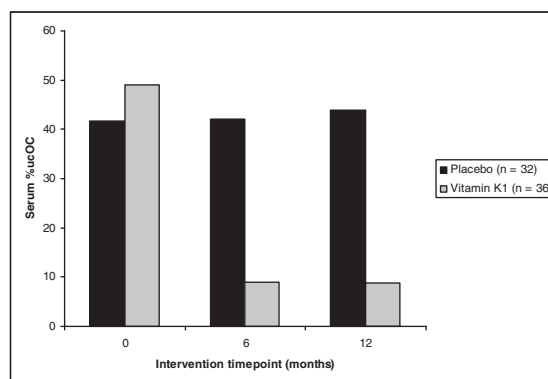
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Adult patients with Crohn’s disease (CD), even those in remission, have been shown to have higher circulating under- γ -carboxylated osteocalcin (ucOC) concentrations, a sensitive marker of vitamin K nutritional status⁽¹⁾, compared to age- and sex-matched healthy control subjects^(2,3). Increased concentrations of ucOC in CD patients in these studies appear to be positively and negatively associated with the rate of bone turnover⁽³⁾ and bone mineral density (BMD) at some sites⁽²⁾, respectively. The aim of our study was to investigate whether supplementation with vitamin K₁ (1000 μ g/d) for 12 months had a positive effect on the rate of bone turnover and BMD in CD patients. We have previously shown that this level of supplementation maximally suppresses the degree of ucOC in CD patients⁽⁴⁾.

Eighty-six adult CD patients, with longstanding disease and in clinical remission at time of inclusion, were recruited and randomised to one of two treatment groups (0 (placebo) or 1000 μ g vitamin K₁/d) along with Ca (500 mg/d) plus vitamin D (10 μ g/d) (to prevent underlying deficiencies of both nutrients) for 12 months. Serum ucOC, γ -carboxylated osteocalcin and %ucOC (indices of vitamin K status), 25-hydroxyvitamin D, parathyroid hormone and markers of bone turnover (serum C-terminal telopeptides of type I collagen, serum bone-specific alkaline phosphatase and urinary N-terminal cross-linked telopeptide of type I collagen) were measured by ELISA. BMD of lumbar spine, femur and ultra-distal radius were measured by dual-energy X-ray absorptiometry at baseline and endpoint. A food frequency questionnaire was used to determine habitual dietary Ca, and vitamins D and K₁.

There were no significant differences in baseline dietary intake, anthropometric measures, biochemical markers of vitamin K status, or in bone turnover or BMD between the two treatment groups. Overall compliance was 90%, with no significant difference ($P>0.8$) between the two intervention groups. All indices of vitamin K status were favourably altered by vitamin K₁ supplementation. Repeated measures ANOVA showed a significant ($P<0.0001$) reduction in serum %ucOC in the vitamin K₁ but not placebo group (see Figure).



There was no significant effect ($P>0.2$) of 12-month supplementation with vitamin K₁ (1000 μ g/d), in addition to Ca and vitamin D, on any of the bone turnover markers or on BMD at any of the skeletal sites, whether unadjusted or adjusted for potential confounding factors such as age, sex, steroid use, site of CD, smoking between the two treatment groups.

In conclusion, despite the fact that 1000 μ g vitamin K₁/d significantly improved vitamin K status in adult CD patients with longstanding disease and currently in remission, there was no apparent benefit to bone health outcomes. Therefore, consideration of the inclusion of vitamin K₁ supplementation to existing dietary guidelines for prevention of osteoporosis in adult CD requires further research justification.

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